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Resource Availability, Mortality and Fertility: A Path Analytic Approach to Global Life History Variation

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Running Title: Resources, Mortality and Fertility

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Abstract: Humans exhibit considerable diversity in timing and rate of reproduction. Life history theory suggests that ecological cues of resource richness and survival probabilities shape human phenotypes across populations. Populations experiencing high extrinsic mortality due to uncertainty in resources should exhibit faster life histories. Here we use a path analytic approach informed by life history theory to model the multiple pathways between resources, mortality rates, and reproductive behavior in 191 countries. Resources that account for the most variance in population mortality rates are predicted to explain the most variance in total fertility rates. Results indicate that resources (e.g., calories, sanitation, education, and health care expenditures) influence fertility rates in paths through communicable and non-communicable diseases. Paths acting through communicable disease are more strongly associated with fertility than are paths through non-communicable diseases. These results suggest that a path analytic approach may help disaggregate extrinsic and intrinsic mortality factors in cross-cultural analyses. Such knowledge may be useful in developing targeted policies to decrease teenage pregnancy and total fertility rates and so issues associated with overpopulation.

Introduction

Human populations exhibit considerable variation in timing and frequency of reproduction. Adolescent fertility rates in Niger, for example, were over 31 times higher than in South Korea (World Health Organization, 2009). Variation in fertility rates across populations result in part from differences in sources of mortality (Roff, 2002; Stearns, 1992). Sources of mortality may respond differently to social resources (access to health care, water and sanitation services; education; income equality etc.). Here we test hypotheses from life history theory concerning the nature of mortality (intrinsic vs. extrinsic) and mortality effects on reproduction. In general, life history theory predicts that high mortality rates cue fast life histories characterized by early reproduction and relatively low parental investment per offspring (Borgerhoff Mulder, 1992; Bulled & Sosis, 2010; Chisholm, 1993; Low, Hazel, Parker, & Welch, 2008; Nettle, 2010; Promislow & Harvey, 1990; Quinlan, 2010; Roff, 2002; Stearns, 1992). A key feature of life history theory, however, divides mortality causes into intrinsic and extrinsic components. Those components of mortality have proved to be exceedingly difficult to isolate empirically. Here we use a path analytic approach to untangle relations between resources, disease, mortality and fertility. This approach allows us to begin to assess effects of extrinsic and intrinsic components of mortality (and the resources associated with each) on human reproductive behavior.

Life History Theory

Life history theory (LHT) provides an evolutionary framework for understanding how environmental cues of resource richness and organismal survivorship affect reproductive decisions. Theoretically, the evolution and development of life history strategies trend towards enhancing individual reproductive fitness in specific environments (Roff, 2002; Stearns, 1992).

Adaptive life history strategies develop, in part, in response to costs and benefits of allocating energy (i.e., resources) to growth, maintenance, and reproduction within variable ecological contexts. As resources invested into one life function (mating) cannot be devoted to another (growth) trade-offs arise (Stearns, 1989).

One of the most fundamental trade-offs in an organism's life-history is between current versus future reproduction (see review in Ellis, Figueredo, Brumbach, & Schlomer, 2009). Fitness costs and benefits are guided by variation in life-expectancy and quality versus quantity of offspring. Delaying reproduction allows an organism to allocate more resources to somatic effort (i.e., growth and maintenance), thereby lengthening life expectancy and increasing the ability to produce and invest in higher quality offspring. This delay, by decreasing energy devoted to reproductive effort, lowers the quantity of offspring across the reproductive lifespan. In contrast, earlier investment in reproduction increases the quantity of potential offspring across the reproductive lifespan but shortens remaining life expectancy when maternal somatic resources are depleted through repeated pregnancy and lactation. Earlier and more frequent investment in reproduction, by limiting somatic investment, decreases the quality of offspring by reducing the amount of parental investment per offspring (Roff, 2002; Stearns, 1992). There is a potentially complex relationship between the allocation of energy to somatic and reproductive effort and life expectancy. Over the life course increased investment in somatic effort should lengthen life span relative to other allocation decisions within a population. However, a longitudinal study of mortality and reproduction indicated that population mortality rates in early life had a causal role in the allocation of somatic and reproductive effort later in adulthood, when early life and later population mortality rates were uncorrelated (Quinlan 2010).

Environmental risk and life history

In life history theory (LHT) the local nature of risk is a major factor affecting trade-offs in the allocation of effort to somatic maintenance, development, mating and parenting (Chisholm, 1999; Quinlan, 2007, 2010). LHT partitions risk into two types: extrinsic and intrinsic. Extrinsic mortality is the risk of death that is not conditional on an organism's reproductive behavior (Stearns 1992:182). Statistically, we can define extrinsic mortality as variance in the probability of death that is not accounted for by mating effort or parenting effort (or by extension tradeoffs between reproductive and somatic effort). In other words, an organism cannot escape extrinsic mortality by changing its behavior: it is the age-specific risk of death that is equally shared by all members of a population. Intrinsic mortality, in contrast, is the probability of death associated with allocation of somatic and reproductive effort. Predation, for example, could be either extrinsic or intrinsic mortality or both. Imagine a population of organisms in which there is a probability (p) of death from predation at age x. Then p is a combination of factors, some are beyond an individual's control but others are not. The frequency by which an individual encounters a predator depends on extrinsic factors such as the density of predators in the environment (beyond the individual's control) *and* intrinsic factors such the level of vigilance, time spent exposed in the landscape as a result of mating effort, etc. (determined by allocation of effort). An individual of a prey species in an environment with many predators may reduce the probability of death by predation by adjusting its behavior, but there is always some extrinsic probability of death by predation. The predation example raises an important point about extrinsic mortality: Any age-specific probability of death has both intrinsic and extrinsic components that can be difficult to isolate analytically. Despite empirical challenges, extrinsic and intrinsic components of mortality can have profound influences on adaptive behavior.

Extrinsic mortality plays a key role in the evolution of life histories and reproductive strategies (Chisholm 1993, 1999; Promislow & Harvey, 1990; Stearns, 1992; Roff, 2002;). When extrinsic mortality is high, then organisms should reproduce early in life to reduce mortality exposure over time and extend the length of the reproductive span, which should maximize fertility to "beat the odds" that some offspring will die. Conversely when extrinsic mortality is low, then differential reproductive success is contingent on resources invested in growth, development and parental effort rather than luck. Hence, in low extrinsic risk environments individuals may enhance fitness by delaying reproduction to accrue additional resources (including knowledge and skills), and by reducing fertility and increasing investment per offspring. Conversely, in high risk environments early reproduction and minimal parental investment per offspring can be adaptive. These predicted relationships hold among mammals: Juvenile mortality is negatively correlated with age at maturity, age at weaning, maternal investment, and positively correlated with litter size, and pace of reproduction (Promislow & Harvey, 1990).

Extrinsic risk for humans has attracted theoretical interest since the early 1990's (e.g., Borgerhoff Mulder, 1992; Chisholm, 1993, 1999; Harpending, Draper, & Pennington, 1990) however, empirical work is relatively scarce. Several studies show predicted relations between extrinsic risk and human life history patterns. Mortality was negatively associated with age at reproductive maturity among urban Americans (Wilson & Daly, 1997), Sub-Saharan Africans (Gant, Heath, Ejikeme, Snell, & Briar-Lawson, 2009) and in four cross-cultural studies (Bulled & Sosis, 2010; Low et al., 2008; Placek & Quinlan, 2011; Walker et al., 2006). Nettle (2010) documented similar relationships across British neighborhoods where economically marginalized (i.e., lower resource availability) neighborhoods displayed earlier ages at reproduction, lower

birth weights, and shorter duration of breastfeeding (see also Nettle, Coall, & Dickins, 2011). In a longitudinal study of a rural Dominican community, Quinlan (2010) found that high infant mortality rates predicted earlier ages of first reproduction, although very high infant mortality rates produced a saturation point of parental investment resulting in reproductive delays. Support for a relationship between mortality rates and life history strategies has also been documented among hunter-gatherer and small-scale horticulturalist groups (Walker et al., 2006). And even perception of mortality may influence human reproductive behavior (Chisholm, Quinlivan, Petersen, & Coall, 2005). This small body of research makes clear that local extrinsic risk is an important environmental cue for shaping human reproductive strategies, but how and when are local environmental conditions encoded into life histories? How do we empirically distinguish between extrinsic and intrinsic components of mortality? Here we use a path analytic approach to identify specific factors mediating and moderating effects on total fertility rates across 191 nations.

Although the distinction between extrinsic and intrinsic mortality is critical for LHT predictions, partitioning mortality into extrinsic or intrinsic components has proved very difficult (Ellis, et al., 2009). In many studies mortality rates are commonly quantified with "all-cause" mortality parameters, such as life expectancy at birth (LEB) or parameters exhibiting strong correlations with LEB, such as infant mortality (see Anderson, 2010; Bulled & Sosis, 2010; Low, et al., 2008; Wilson & Daly, 1997). Even studies using "all-cause" mortality measures across and within taxa have demonstrated a strong positive relationship between higher rates of mortality and faster life history strategies. Better predictive models and theoretical development await improved analytical strategies that can identify components of mortality.

Resources and life history

Studies across and within human populations, in agreement with findings across numerous nonhuman species, indicate that relatively higher mortality rates are associated with both earlier onset and higher rate of reproduction(Anderson, 2010; Bulled & Sosis, 2010; Low, et al., 2008; Promislow & Harvey, 1990; Quinlan, 2010; Stearns, 1992). Unlike any other species however, humans are capable of producing resources that lead to increases and decreases in survival probabilities of mortality causes and thus may play a direct role in population mortality variation. For example, access to medicinal resources can increase the survival probability of certain diseases while weapons of modern warfare can decrease the survival probability of conflict. This capacity is important in a life history framework as resources may transform an extrinsic cause of mortality to an intrinsic cause. For instance, malaria may be defined as a source of extrinsic risk when individuals lack access to necessary medication or preventative measures. When medicines/preventative measures become available however, somatic investment (e.g., searching for employment in order to afford medicine) can increase the survival probability associated with malaria thus making malaria an intrinsic cause of morality. Theoretically, we expect access to malaria medicines/preventative measures will lead to increases in a population's LEB and thus alter the influence of mortality from malaria on fertility. Critically, a cause of mortality previously associated with "faster" life history strategies now cues development of "slower" strategies.

Previous studies examining the relationship between resources and life history strategies in humans have primarily focused on indirect proxies of resource availability, such as participation in education/workforce, and their associations with mortality (Bulled & Sosis, 2010; Low, et al., 2008; Wilson & Daly, 1997). Theoretically, investment in education and employment, by representing an increase in somatic investment, should coincide with delays in

reproduction and increases in LEB. Further, participation in education/workforce should increase as population mortality rates fall thereby increasing the probability that future benefits of an education and employment will be accrued (Hill & Kaplan, 1999; Kaplan, Hill, Lancaster, & Hurtado, 2000). Empirical support for a relationship between investment in education/workforce and longer LEB has found some support across cultures. Low et al. (2008) documented a significant moderate to strong correlation between LEB and female secondary school enrollment of (r = .405, $p<.05$). However, female participation in the workforce did not have a significant correlation with LEB. This non-significant effect may arise given that a large majority of females in developing countries with comparatively low LEB are employed in the agricultural sector (Low, 2008). Bulled and Sosis (2010) documented a similar relationship with LEB displaying a strong positive relationship with Adult Literacy Rate (r =.699, $p<.01$) overall school enrollment (r=.753, $p<.01$) secondary school enrollment (r=.810, $p<.01$) and tertiary school enrollment (r=.676, $p<.01$). However, there was not a significant relationship between LEB and primary school enrollment (r=.103, $p>.05$). The authors suggest this non-significant effect indicates that a threshold of educational attainment must be reached (i.e., secondary) before effects on LEB are noticeable.

Predictions

The current paper examines the tradeoff between current and future reproduction in 191countries by testing two hypotheses about the onset and frequency of female reproduction. Female fertility in the 15-19 cohort is used as proxy for early reproduction. Frequency of reproduction in females is represented by total fertility rates. Differences in adolescent and total fertility rates across nations reflect variation in life history strategies on the fast to slow spectrum with earlier reproduction and higher rates indicating faster life history strategies and later

reproduction and lower rates indicating slower strategies. The first hypothesis tested is that *causes of mortality with the greatest impact on population mortality rates will have the largest impact on adolescent and total fertility rates*. Causes of mortality with the greatest impact on population mortality rates include those that impact survival associated with younger age cohorts, because mortality rates in younger cohorts have a greater relative impact on LEB than older cohorts. Mortality causes that preferentially impact younger age cohorts, especially in children under five, are often communicable diseases (e.g. HIV, malaria, pneumonia) (Leowski, 1986; Lopez, Mathers, Ezzati, Jamison, & Murray, 2006; Sachs & Malaney, 2002). Hence we predict that *mortality attributable to communicable diseases account for more variance in adolescent and total fertility rates than non-communicable diseases*. Beyond differences in transmission, communicable diseases (e.g., malaria, tropical cluster diseases) exhibit a larger impact on younger-age cohorts, especially infants, whereas many non-communicable diseases (e.g., cancer, type 2 diabetes) have greater impacts on older cohorts. Building upon this, the second hypothesis tested is that *resources with the greatest impact on the survival probabilities of communicable diseases will have the greatest impact on adolescent and total fertility rates.* Resources affecting survival probabilities of communicable diseases are those affecting transmission environments and the availability of healthcare (e.g., medicine and preventative measures) (Watson, Gayer, & Connolly, 2007) . Based on this reasoning we predict *resources affecting the transmission environment and the availability of treatment and preventative measures will have the largest impact on the survival probabilities associated with communicable diseases*.

Materials and Methods

Data Analysis

A path analytic (PA) approach was used to model the relationships between resources,

mortality parameters and total fertility rates. PA is an extension of multiple regression where regression is conducted over a set of variables. Results of a PA, called "path coefficients", reflect the magnitude and statistical significance of the predicted relationships across the set of variables. PA has a number of analytical strengths compared to the ordinary least squares (OLS) regression techniques used in previous studies (e.g., Bulled & Sosis 2010; Low et al. 2008; Wilson & Daly 1997). Most important among these is the ability to precisely specify the form and complexity of life history relationships. Path models can specify the causal relationships predicted to operate between resources, morality and life history strategies. Enabling this specification is the use of mediator variables, which act as both dependent and independent variables. As both dependent and independent variables, mediator variables allow for the quantification of the indirect relationships, referred to as indirect effects, which are predicted to exist between a set of variables (e.g., resources, mortality, and fertility). Calculation of indirect effects allows for more nuanced tests of life history predictions because the effect of resources on life history strategies is likely mediated through a resource's prior impact on population mortality rates. For example, access to clean water, while it may not directly impact total fertility rates, indirectly impacts these rates through prior direct effects on mediator variables that *do* have direct effects on total fertility rates, such as population mortality rates. Indirect effects are calculated as the product of the direct effects. Both direct and indirect effects are interpreted as regression coefficients.

Data Sources

Data used in the analysis was gathered from several online databases at the UN Data portal (http://data.un.org) on 191 United Nations countries. Resource variables represent data from years 1999-2003, causes of mortality variables are taken from 2004, LEB from 2005, and

fertility data from 2007. It would have been preferable to use resource variables collected in the same year. However, given the number of resource variables in the model and data for resources are not collected every year for every country, it was not possible to find a year in which all resource variables were collected. Data primarily derive from civil registration records, and/or surveys and censuses. Variable descriptions, labels, years, and data sources are provided in Table 1.

Data definitions

Fertility rate indicators included in the models were total fertility rate and age-specific fertility for females 15 to 19 years old. Total fertility rate (*TFR*) is the average number of births expected across a female's reproductive life-span if current age-specific fertility rates remained constant. Age specific fertility (*ASF*) is defined as the number of births per 1000 women in a given age range. Mortality rate indicators included in the model were life expectancy at birth (*LEB*) and years lost to communicable (*comm*) and non-communicable diseases (*noncom*). Years lost to communicable diseases reflect a percentage of the distribution of years of life lost to communicable disease. Years lost to non-communicable disease are age-standardized mortality rates for non-communicable diseases. A complete list of the diseases included in the calculation of years lost to communicable and non-communicable diseases can be accessed at http://www.who.int. Resource indicators included in the model were access to clean water and sanitation services (*clean*), total healthcare expenditure (*health*), calories per capita (*calorie*), GINI (*GINI*), adult female literacy (*femlit*), and contraception prevalence rate (*CPR*). Access to clean water and sanitation is a percentage reflecting the proportion of the population using improved drinking-water and sanitation facilities. Percentages of access to clean water and sanitation were combined into a composite variable reflecting the proportion of the population

with access to both clean water and sanitation facilities. Total expenditure on healthcare reflects the per capita expenditure from both government and non-governmental agencies on health care services. Per capita values are in US dollars and are based off the purchasing power parity. Years lost to communicable diseases reflect a percentage of the distribution of years of life lost to communicable disease. The GINI coefficient is an indicator of income inequality where a score of 0 indicates complete equality and 1 indicting complete inequality in income. Data on adult female literacy rates reflect females age 15 and above. Contraceptive prevalence rate includes both modern and traditional methods.

All analysis in were done in M*plus* (Version 6.1,Muthén & Muthén, 2010) and Stata 11 (StataCorp, 2009). As a previous study by Quinlan (2010) documented a quadratic relationship between mortality and age at first birth, quadratic effects between mortality parameters (i.e., LEB and mortality causes) and fertility parameters (i.e., adolescent and total fertility) was modeled but were not significant. Likewise, the potential for interaction affects among resource variables were tested but did not result in a better fitting model. Several variables were missing data from a few countries. A benefit of M*plus* is that it uses a full-information maximum likelihood estimator which uses all available data, (i.e., N = total sample size), including cases with missing data (Brown, 2006). Although the amount of missing data was small, a description of the missing data is provided in covariance coverage matrices in Appendix C. Due to significant levels of skewness and kurtosis in some variables (see Table 2) a maximum likelihood estimator with robust standard errors (MLR) was used. Correlations among the variables are provided in Table 3.

Results

Model Fit

MLR estimation converged on an admissible solution for both path models. Global and localized fit indices indicate both models displayed good overall fit (see Table 4). Model chi-squares were non-significant, Model 1: $X^2_M = 17.62$ (p=.309, df_M=15), Model 2: $X^2_M = 3.90$ (p=.79, df_M=7) and so the exact-fit hypothesis, (i.e., no discrepancies between population and model predicted matrix) cannot be rejected (Kline, 2010). The Standardized Root Mean Square Residual (SRMR) value, which can be conceptualized as the average discrepancy between the correlations in the matrix of observed values and those in the model predicted matrix, were below the suggested .08 value for both models (Brown, 2006). For Model 2, the Root Mean Square Error of Approximation (RMSEA) and the associated 90% C.I. were below the suggested .06 cut-off criteria (Hu & Bentler, 1999). For Model 1, the upper level of the 90% C.I. for the RMSEA was above the suggested .06 cut-off criteria but still below .08, which is consistent with a mediocre model fit (MacCallum, Browne, & Sugawara, 1996). Hu and Bentler (1999), however, note that the RMSEA test tends to over-reject models with small sample sizes, which characterizes the current sample *(n*=191). Evaluation of model fit through comparative fit indices, which compare the model to a more restricted or "parsimonious" model provide further evidence of good fit for both path models. Both the Comparative Fit Index (CFI) and the Tucker Lewis Index (TLI) values were above the suggested .95 cut-off criteria (Hu & Bentler, 1999).

Localized fit indices indicated good overall fit for both path models. For Model 2, inspection of modification indices (MI), which indicate the increase in model X^2, revealed no areas of localized ill-fit. Inspection of the MI for Model 1 revealed no areas of ill-fit with the exception of two parameters, a direct path between *ASF* and *clean* (MI = 4.545) and a correlation between *femlit* and *LEB* (M.I. = 4.635). Inclusion of a direct path from *ASF* to *clean* and a correlation between *femlit* and *LEB* were not significant ($p>.05$) and so were not included in the

model. Further evidence of good localized fit was displayed in the standardized residuals, which reflect how well the variances and covariance matrix produced by the model parameters fit the observed variance and covariance matrix. Standardized residuals, which are interpreted as *z*-scores, can be conceived as the number of standard deviations by which the predicted residuals differ from zero-value residuals that would result from a perfectly fitting model (Brown, 2006). For Model 2, there were no residuals above the 2.58 significance level. For Model 1, the sole standardized residual above the 2.58 significance level was a negative residual (-2.78) between *noncomm* and *ASF*. As this residual is negative, it indicates that the model parameters overestimate the observed relationship between *noncomm* and *ASF.* Although significant, this residual is not an outlying value, which may have been indicative of serious model misspecification, as other residuals are close to the 2.58 cut-off point, (Brown, 2006).

Model Interpretation

Path diagrams representing the predicted relationship between resources, mortality, and fertility are presented in Figures 1 and 2. These figures can be conceptualized as the graphical equivalent of a set of regression equations that relate the dependent and predictor variables (Byrne, 2012). Each path tested is indicated by a straight line with a single-headed arrow, which points in the proposed direction of causality. Path coefficients (the number immediately above or below the single headed arrow) are standardized and are interpreted as the expected change in standard deviation units of the dependent variable given a one standard deviation change in the predictor variable, controlling for the direct effects of other variables. The curved double headed arrows on the left side of the model indicate correlations between pairs of predictor variables. The strength of the correlation between two variables is indicated by the number within the curved double-headed arrow connecting those two variables. The number inside the circles adjacent to each dependent variable indicates the residual variance associated with that

dependent variable.

Interpretation of the path coefficients will follow the predicted relationships between resources, mortality, and fertility rates. Predictors of mortality causes will be discussed first followed by predictors of LEB (for Model 1), and finally predictors of adolescent and total fertility. Direct effects on a dependent variable are discussed before discussion of the indirect effects (see "Data Analysis" section for explanation of direct and indirect effects). Standardized path coefficients predicting years lost to communicable diseases, LEB, and adolescent and total fertility rates are translated into original metrics. Adolescent fertility rates are rounded up to the next birth.

Results: Model 1

Mortality Causes

Resource variables, accounted for 84.2% of the variance in years lost to communicable disease and 58.9% of the variance in years lost to non-communicable disease (see Table 5). Access to clean water and sanitation services had a strong effect on years lost to communicable diseases with a one *sd* increase predicting a 13.7 decrease in years lost controlling for other resources. Remaining resource variables (i.e, *calories, GINI, CPR, femlit)* exhibited similar affects with *sd* increases resulting in an approximate 4 year decrease in years lost to communicable disease (see Table 4). The sole exception to this trend was total healthcare expenditure, which did not account for a significant portion of variance in years lost to communicable diseases *(p* >.05). Total healthcare expenditure, however, exhibited the strongest effect on years lost to non-communicable diseases, with a one *sd* increase resulting in a -.45 *sd* decrease in years lost. Calories per capita and contraception use had similar impacts on years lost to non-communicable diseases with *sd* increases predicting an approximate .25 *sd* decrease.

GINI exhibited the smallest effect with a *sd* increase (more inequality) resulting in a .16 *sd* decrease in years lost to non-communicable diseases.

Life Expectancy at Birth

Summed across direct and indirect effects, resource variables and mortality causes accounted for 88.6% of the variance in LEB (see Table 5). Significant direct effects on LEB were produced through total healthcare expenditure and years lost to communicable and non-communicable diseases One *sd* increases in years lost to communicable and non-communicable diseases predicted an 8.40 year and a 3.16 year decrease in LEB, respectively. Total healthcare expenditure exhibited the smallest direct effect on LEB predicting a .75 year decrease. Remaining resource variables had indirect effects on LEB through prior direct effects on years lost to communicable and/or non-communicable diseases. Access to clean water and sanitation services exhibited the largest indirect effect on LEB with a one *sd* increase predicting a 4.10 year increase in LEB. *Sd* increases in other resource variables had similar but smaller impacts on LEB with calories per capita and contraception prevalence rate predicting an approximate 2 year increase and total healthcare expenditure an approximate 1 year increase in LEB.

Adolescent fertility rates

The final model accounted for 60% of the variance in adolescent fertility rates (see Table 5). Variables with a direct effect on adolescent fertility rates were LEB, the GINI coefficient and adult female literacy rates. LEB had the strongest direct effect on adolescent fertility rates with every *sd* increase associated with a 19 birth decrease per 1000 adolescent women. The GINI coefficient and adult female literacy rate also had indirect effects on adolescent fertility rate through prior direct effects on years lost to communicable and/or non-communicable diseases. Summed across both direct and indirect effects, one *sd* increases in female literacy rates and

GINI predicted 15 and 12 birth decreases, respectively. The impacts of mortality causes (i.e., communicable and non-communicable diseases) on adolescent fertility were completely mediated through prior direct effects on LEB. A one *sd* increase in years lost to communicable diseases predicted a 15 birth decrease while a smaller indirect effect was produced by years lost to non-communicable disease with a one *sd* increase predicting a 6 birth decrease. All resource variables had an indirect effect on adolescent fertility rates. The strongest indirect effect on adolescent fertility rate was produced through access to clean water and sanitation with a one *sd* increase predicting a 7 birth decrease. Calories per capita and contraception prevalence rate predicted approximately a 4 birth decrease, respectively. Total healthcare expenditure had the smallest effect on adolescent fertility rates predicting a 3 birth decrease.

Results: Model 2

Mortality Causes

Resource variables accounted for 82% of the variance in years lost to communicable and 58% of the variance in years lost to non-communicable disease (see Table 6). In general, both the pattern and magnitude of relationships found between resources and mortality causes were similar to Model 1. Access to clean water and sanitation services had the strongest direct effect on years lost to communicable diseases with a one *sd* increase predicting a 9.6 decrease in years lost, controlling for the direct effect of other resources. Calories per person and adult female literacy rates had a smaller but similar direct effect with *sd* increases resulting in an approximate 6 year decrease in years lost to communicable disease. Income inequality and contraception prevalence rates exhibited the smallest direct effects with a one s*d* increases in each predicting a 5.1 year increase and 4.3 year decrease in years lost to communicable diseases, respectively. Like Model 1, the sole resource variable without a significant direct effect on years lost to

communicable diseases was total healthcare expenditure. Total healthcare expenditure however, exhibited the strongest effect on years lost to non-communicable diseases with a one *sd* increase resulting in a -.46 *sd* decrease in years lost. Contraception prevalence rate had the second largest direct effect with a *sd* increase predicting a -.281 *sd* decrease in years lost to non- communicable disease. Calories per person exhibited the third largest effect on years lost with a *sd* increase predicting a -.210 *sd* decrease. Income inequality exhibited the smallest direct effect on years lost to non-communicable diseases with a *sd* increase predicting a -.156 *sd* decrease.

Life expectancy at birth was not included in Model 2 as its effect on total fertility rate was completely mediated by years lost to communicable and non-communicable disease. Additionally, its inclusion did not improve the global or local fit of Model 2. Due to the absence of LEB both years lost to communicable and non-communicable diseases had direct effects on TFR.

Total fertility rates

The final model accounted for 76% of the variance in total fertility rates across 191 nations (see Table 6). Again, both the pattern and magnitude of relationships found between predictor variables and fertility were consistent with results from Model 1 Variables with a direct effect on total fertility rates were the mortality variables of years lost to communicable and non-communicable disease and the resource variables of total healthcare expenditure, contraception prevalence rate, and adult female literacy rate. Across all variables, years lost to communicable disease had the largest impact with a one *sd* increase predicting a .82 increase in fertility across the reproductive lifespan. Contraception prevalence rate exhibited the second largest direct effect with a *sd* increase predicting a .41 decrease in TFR. The direct effect of adult female literacy, which was approaching significance (*p=.077),* predicted a .22 decrease in TFR for every *sd*

increase. The effect of total healthcare expenditure was of similar magnitude but in the opposing direction with a *sd* increase predicting a .21 increase in TFR. Years lost to non-communicable disease had the smallest direct effect on TFR, with a one *sd* increase predicting a .19 increase in fertility.

All resource variables, including those with direct effects, had indirect effects on TFR. Indirect effects were produced through a resources prior direct effect on years lost to communicable and/or non-communicable diseases. Like Model 1, the largest indirect effect on total fertility rate was exhibited by access to clean water and sanitation services with a one *sd* increase predicting a .28 decrease in TFR. Calories per person had the second largest indirect effect with a *sd* increase predicting a .24 decrease in TFR. A *sd* increase in adult female literacy rates predicted a .19 decrease in TFR. The indirect effects of contraception prevalence rate and income inequality were similar in magnitude but in the opposing direction with a *sd* increase predicting a .13 decrease and a .12 increase, respectively. Total healthcare expenditure had the smallest indirect effect on total fertility rates with a *sd* increase predicting a .09 decrease in fertility across the female reproductive lifespan

Discussion

Path analysis allows us to begin partitioning mortality into extrinsic and intrinsic components – a crucial next step in human life history research. Results from both path models provide strong support for theoretical predictions and largely concur with results of previous studies. Higher population mortality rates, as reflected by lower life expectancy at birth (Model 1) and greater years lost to communicable and non-communicable diseases (Model 2), are associated with “faster” life history strategies, as indicated by higher adolescent and total fertility rates. For example, as indicated by Model 1, every year decrease in LEB predicts two more

births per 1000 adolescent females Numerous studies have documented this relationship between mortality and fertility; however, the current study models how the availability of resources, through prior impacts on mortality, ultimately affect the timing and frequency of reproduction in humans. In particular, the use of a path analytic approach enables the test of whether decreases in resources, by mediating an individual's ability to cope with mortality causes, lead to faster reproductive strategies. Both models supported this relationship. In Model 1, a standard deviation decrease in all resources, including access to education, healthcare, clean water and sanitation, calories, contraception and income equality (i.e., GINI) predicted a one standard deviation increase in adolescent fertility ($sd \approx 44$). Similarly, in Model 2, a standard deviation decrease in access to education, clean water and sanitation, calories, and income equality combined to predict 1.58 more births across the female reproductive life-span, slightly more than one standard deviation. While both path models generate a more nuanced representation of the relationship between resources and fertility rates they also allow for the quantification of the relative impacts associated with each resource. A resource's relative impact is calculated by division of the standardized total effects (see Tables 5 and 6). In Model 1, for example, division of the total effects of female literacy (-.388) and total healthcare expenditure (-.027) on adolescent fertility reveals that female literacy has a 14 times greater impact on adolescent fertility rates.

Mortality causes accounting for the most variance in population mortality rates account for the most variance in adolescent fertility rates. Communicable diseases account for more of the variance in both LEB and fertility rates. Model 1 indicates that every standard deviation increase in years lost to communicable disease exhibited an almost three times greater impact on LEB (8.40 yr) compared to non-communicable diseases (3.16 yr). Effects of mortality causes on

fertility rates were completely mediated by LEB in Model 1. Model 2 also shows that communicable diseases account for more variance in total fertility rates.

Results of both models also supported the second hypothesis: Resources impacting the survival probabilities of communicable diseases – by impacting transmission, prevention and treatment –have stronger effects on fertility than resources impacting survival probabilities of non-communicable diseases. Access to clean water and sanitation showed the largest indirect effect and third largest total effect on fertility. Contraception prevalence rate had the third largest indirect effect of all resource variables on adolescent fertility rates. Adult female literacy rates, which exhibited the third largest indirect effect on total fertility in Model 2, indirectly impact the disease transmission environment as literate females may be more educated in disease prevention (e.g., sex education).

Our results, in general, concur with predictions from life history theory and previous studies. However, a few predicted relationships were not found. In both models total health care expenditure did not have a significant impact on years lost to communicable disease. This finding may indicate that total healthcare expenditure is not a strong indicator of access to health services that specifically target communicable diseases, or a majority of healthcare funds are allocated to the treatment of non-communicable diseases. In the majority of developed nations communicable diseases with the potentially greatest impact on life expectancy(i.e., diseases that affect childhood mortality) have either been eradicated through large-scale immunization programs (e.g., typhoid, cholera, and tuberculosis) or by tactics and infrastructure improvements that decrease transmission rates (e.g., mosquito prevention programs). As a result of these measures the mortality rates in developed countries are less impacted by communicable diseases (World Health Organization, 2009). Extension of adult life expectancy increases age-related non-

communicable diseases (e.g., cancer, and heart disease) that are expensive to treat (Shelton, 2007), the strong relationship between total healthcare expenditure and non-communicable diseases in developed countries may disguise the effect of total healthcare expenditure on communicable diseases in developing countries. Alternatively, healthcare funding in developing countries may not have yet to produce a significant decrease in mortality from communicable diseases for many years. This reasoning may partly explain the unexpected result in Model 2 where total healthcare expenditure predicted an increase in total fertility rates. Future studies should incorporate indicators of healthcare funding with direct relationships to communicable diseases (e.g., access to immunization programs and STI prevention education).

Some resources, including adult female literacy rate, contraception prevalence rate, and income inequality affect fertility rates largely outside the context of mortality. Female literacy and the income inequality produced the smallest indirect effects on life expectancy in model 1, but they were the only resources variables with a direct effect on adolescent fertility. Effects of education and income inequality on life expectancy may need to be more closely evaluated. Another possibility may be that a threshold level of education must be reached before effects on LEB are statistically noticeable (Bulled and Sosis 2010). More difficult to explain is the weak effect of income inequality on LEB. The likely consequences of income inequality on access to healthcare and overall standard of living suggest that variation in population LEB should be intimately tied with the GINI coefficient. However, inequality may interact with other variables in complex ways not detectable in a global comparison.

In sum, on a global scale resources influence life history largely through their impact on communicable diseases. However, in populations where disease burdens have been substantially reduced, then other indicators of extrinsic and intrinsic risk come into play. A promising line of

research indicates that in healthy populations, psychosocial stress apparently tunes life history development in ways similar to mortality in less developed populations (Chisholm & Coall, 2008).

Conclusion

Data from 191 countries were used to test two hypotheses operating as separate links in the causal chain from resources to fertility rates. Results of two path models confirm that resources with the greatest impact on the survival associated with communicable diseases have the greatest impact on the timing and frequency of reproduction. A path analytic approach generates a more nuanced representation of the direct and indirect relationships operating between resources and reproductive behavior. This approach suggests that some environmental factors, such as communicable versus non-communicable disease, appear to have effects more like extrinsic risks versus intrinsic risks. While this study does not entirely resolve important issues in isolating mortality sources, it does improve our understanding of local conditions' influence on life history strategies. Hence, this approach may prove useful in new theory development and in population planning.

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Table 1. Variable labels, years and sources.

variable	*label*	*year(s)*	*Source*
total fertility rate	*TFR*	2007	World Health Organization
age specific fertility 15-19 y.o.	*ASF*	2007	World Development Indicator
life expectancy at birth	*LEB*	2005	World Development Indicator
years lost to communicable disease	*comm*	2004	World Health Organization
years lost to non-communicable disease	*noncomm*	2004	World Health Organization
calories per capita	*calorie*	2000-2003	World Health Organization
total healthcare expenditure	*health*	2000	World Health Organization
female literacy rate	*femlit*	2000-2001	Gender Info
GINI	*GINI*	1999-2003	Human Development Report
access to clean water and sanitation	*clean*	2000	World Health Organization
Contraception prevalence rate	*CPR*	1999-2003	State of the World's Children

Table 2. Variable parameters. See Table 1 for explanation of variable label. * significant at $p<.05$

variable	*M*	*min-max*	*Sd*	*skew*	*kurtosis*
TFR	2.94	1.2-7.2	1.53	1.05	3.24
ASF	53.14	3.16-201.41	43.58	.98	3.13
LEB	66.7	41.21-82.10	10.65	-.73	2.41
comm	37.40	31-87	28.08	.42	1.73
noncomm	681.46	284-1309	200.29	.00	2.58
calorie	2689.78	1557-3814	505.83	.16	2.39
health	631.67	1-4570	879.29	2.05*	7.02*
femlit	78.29	12.6-99.9	24.41	-1.07	2.97
GINI	40.86	24.7-74.3	9.42	.50	2.99
clean	74.63	18-100	23.84	-.59	2.06
CPR	47.39	3-96	22.49	-.13	2.00

Table 3. Variable correlations. See Table 1 for explanation of variable label.

	TFR	*ASF*	*LEB*	*comm*	*noncomm*	*calorie*	*health*	*clean*	*GINI*	*femlit*	*CPR*
TFR	1										
ASF	.783	1									
LEB	-781	-.717	1								
comm	.822	.745	-.917	1							
noncomm	.578	.450	-.713	.592	1						
calorie	-.607	-.599	.722	-.733	-.623	1					
health	-.454	-.466	.602	-.601	-.672	.703	1				
clean	-.728	-.718	.836	-.834	-.572	.661	.538	1			
GINI	.276	.431	-.345	.427	.120	-.361	-.372	-.288	1		
femlit	-.735	-.634	.690	-.752	-.509	.499	.463	.755	-.184	1	
CPR	-.754	-.510	.697	-.698	-.576	.533	.444	.677	-.116	.705	1

Table 4. Global fit indices for Model 1 and Model 2.

Index	Model 1 Values	Model 2 Values
X^2_M	17.17	3.90
df_M	15	7
P	.309	.791
RMSEA (90% C.I.)	.027(.000-.076)	.00(.000-.058)
CFI	.998	1.00
TLI	.995	1.01
SRMR	.015	.009

Table 5. Model 1, effect decomposition table. Bolded numbers are the total effects of a variable. See Table 1 for explanation of variable label.

	Dependent			
	COMM	**NONCOMM**	**LEB**	**ASF**
LEB				
Direct Effects				-.436
Indirect Effects				
Total Effects				**-.436**
years lost communicable				
Direct Effects			-.789	
Total Indirect Effects				.344
Total Effects			**-.789**	**.344**
years lost non-communicable				
Direct Effects			**-.**297	
Indirect Effects				.130
Total Effects			**-.297**	**.130**
calories per capita				
Direct Effects	-.184	-.234		
Indirect Effects			.215	-.094
Total Effects	**-.184**	**-.234**	**.215**	**-.094**
total health expenditure				
Direct Effects		-.445	-.071	
Indirect Effects			.132	-.027
Total Effects		**-.445**	**.061**	**-.027**
clean water and sanitation				
Direct Effects	-.488			
Indirect Effects			.385	-.168
Total Effects	**-.488**		**.385**	**-.168**
GINI (Income Inequality)				
Direct Effects	.174	-.157		.228
Indirect Effects			-.090	.039
Total Effects	**.174**	**-.157**	**-.090**	**.287**
adult female literacy rate				
Direct Effects	-.136			-.291
Indirect Effects			.108	-.047
Total Effects	**-.136**		**.108**	**-.338**
contraception prevalence				
Direct Effects	-.144	-.275		
Indirect Effects			.196	-.085
Total Effects	**-.144**	**-.275**	**.196**	**-.085**

Table 6. Model 2, effect decomposition table. Bolded numbers are the total effects of a variable. See Table 1 for explanation of variable label.

	Dependent		
	COMM	**NONCOMM**	**TFR**
years lost to communicable disease			
Direct Effects			.537
Total Indirect Effects			
Total Effects			**.537**
years lost to non-communicable disease			
Direct Effects			.125
Indirect Effects			
Total Effects			**.125**
calories per capita			
Direct Effects	-.244	-.210	
Indirect Effects			-.158
Total Effects	**-.244**	**-.210**	**-.158**
total healthcare expenditure			
Direct Effects		-.457	.136
Indirect Effects			-.057
Total Effects		**-.457**	**.079**
clean water and sanitation			
Direct Effects	-.342		
Indirect Effects			-.184
Total Effects	**-.342**		**-.184**
GINI (Income Inequality)			
Direct Effects	-.180	-.156	
Indirect Effects			.077
Total Effects	**-.180**	**-.156**	**.077**
adult female literacy rate			
Direct Effects	-.229		-.141
Indirect Effects			-.123
Total Effects	**-.229**		**-.264**
contraception prevalence			
Direct Effects	-.154	-.281	-.269
Indirect Effects			-.083
Total Effects	**-.154**	**-.281**	**-.352**

Appendix A. Relationships between indicators of fertility.

Table 7. Correlations among fertility rates (i.e., asf) at every age cohort and total fertility rate (i.e, TFR). Correlations are calculated from raw values.

	asf15_19	asf20_24	asf25_29	asf30_34	asf35_39	asf40_44	asf45_49	TFR
asf15_19	1							
asf20_24	0.85	1						
asf25_29	0.68	0.89	1					
asf30_34	0.62	0.78	0.96	1				
asf35_39	0.63	0.80	0.93	0.98	1			
asf40_44	0.65	0.80	0.89	0.93	0.97	1		
asf45_49	0.54	0.70	0.79	0.81	0.86	0.90	1	
TFR	0.78	0.92	0.96	0.95	0.95	0.94	0.84	1

Appendix B. Relationship between variables and population size of country.

Table 8. Correlations between country population size and variables. Across all variables only contraception prevalence rate has a significant correlation with population size (r =.172, p<.05). Correlations are calculated from raw values. See Table 1 for explanation of variable label.

	TFR	comm	noncomm	calorie	health	clean	GINI	CPR	femlit
population	-0.052	-0.005	-0.024	0.057	-0.027	-0.028	0.091	0.172*	-0.049

Appendix C. Proportion of Data Present.

Table 9. Covariance Coverage Matrix for Model 1. Indicates the proportion of raw data present for each variable and pairs of variables prior to estimation. See Table 1 for description of variable label.

	ASF	LEB	comm	noncomm	calorie	health	clean	GINI	CPR	femlit
ASF	0.921									
LEB	0.916	0.921								
comm	0.916	0.916	0.995							
noncomm	0.916	0.916	0.995	0.995						
calorie	0.859	0.859	0.885	0.885	0.890					
health	0.901	0.901	0.974	0.974	0.874	0.979				
clean	0.874	0.874	0.932	0.932	0.853	0.921	0.937			
GINI	0.874	0.874	0.901	0.901	0.848	0.890	0.864	0.906		
femlit	0.895	0.890	0.916	0.916	0.848	0.901	0.869	0.869	0.921	
CPR	0.796	0.801	0.848	0.848	0.775	0.832	0.806	0.785	0.785	0.853

Appendix C (cont.). Proportion of Data Present.

Table 10. Covariance Coverage Matrix for Model 2. Indicates the proportion of raw data present for each variable and pairs of variables prior to estimation. See Table 1 for description of variable label.

	TFR	comm	noncomm	calorie	health	clean	GINI	CPR	femlit
TFR	1.000								
comm	0.995	0.995							
noncomm	0.995	0.995	0.995						
calorie	0.885	0.880	0.880	0.885					
health	0.979	0.974	0.974	0.869	0.979				
cleansani	0.937	0.932	0.932	0.848	0.921	0.937			
GINI	0.906	0.901	0.901	0.843	0.890	0.864	0.906		
CPR	0.853	0.848	0.848	0.775	0.832	0.806	0.785	0.853	
femlit	0.927	0.916	0.921	0.848	0.901	0.874	0.874	0.791	0.927

Appendix D. Raw Data.

Table 11. Raw Data. See Table 1 for description of variable label.

Country	TFR	ASF	LEB	Comm	Noncomm	Cal	CS	Femlit	GINI	Health	CPR
Afghanistan	7.1	125	43	77	1309	.	25.5	12.6	60	91	10
Albania	2.1	14	76	12	752	2875	93	98.3	33	239	60
Algeria	2.4	8	72	43	565	2928	90.5	60.1	35.3	188	61
Andorra	1.3	.	.	7	373	.	100	99.9	.	1905	.
Angola	6.5	127	46	81	1071	1902	42	54.2	58.6	56	6
Antigua and Barbuda	2.1	.	.	17	674	2378	93	.	57.3	599	53
Argentina	2.3	58	75	18	515	3180	92.5	97.2	50	814	.
Armenia	1.4	36	73	13	1064	2006	91	99.2	33.8	130	53
Australia	1.8	15	80	6	355	3110	100	99	35.2	2271	.
Austria	1.4	13	79	4	409	3794	100	99.9	29.1	2858	51
Azerbaijan	1.8	34	67	37	856	2387	78	98.2	36.5	104	51
Bahamas	2	54	72	36	509	2736	98.5	96.5	57	1361	.
Bahrain	2.3	17	75	12	678	.	.	83.6	.	820	62
Bangladesh	2.9	76	65	61	730	2158	55.5	41.4	33.4	27	56
Barbados	1.5	43	76	22	531	2946	100	99.7	39	916	55
Belarus	1.2	22	69	5	854	2895	96	99.4	29.7	328	73
Belgium	1.6	8	79	5	437	3695	100	99	33	2519	78
Belize	3	81	76	33	677	2867	69	77.1	51	229	34
Benin	5.5	113	60	78	835	2537	44	23.3	36.5	50	17
Bhutan	2.2	43	65	57	708	.	66.5	34	46.8	132	35
Bolivia	3.5	79	65	54	765	2228	60.5	80.7	58.2	282	61
Bosnia and Herzegovina	1.2	17	75	6	670	2723	96.5	94.4	26.2	282	36
Botswana	2.9	54	49	84	594	2256	70	81.8	61	374	48
Brazil	2.3	78	72	30	625	3002	81.5	88.8	55	506	81
Brunei Darussalam	2.3	26	77	16	473	2758	.	90.2	.	1036	.
Bulgaria	1.3	43	73	5	733	2544	99	97.7	29.2	377	86
Burkina Faso	6	132	52	82	924	2439	32.5	15.2	39.6	41	17
Burundi	6.8	21	49	80	919	1604	56.5	52.2	42.4	12	9
Cambodia	3.2	41	58	67	832	2011	27	64.1	40.7	51	40
Cameroon	4.4	129	50	78	840	2254	55	59.8	44.6	75	29
Canada	1.5	13	80	6	374	3178	100	99	32.6	2514	75
Cape Verde	3.4	96	70	53	591	3286	60.5	69.2	50.5	97	61
Central African Republic	4.6	110	44	78	868	1968	42.5	33.5	43.6	25	19

Chad	6.2	169	51	82	910	2083	20.5	12.8	39.8	49	3
Chile	1.9	60	78	10	458	2867	92	95.6	54.9	572	58
China	1.7	10	73	20	627	2979	69.5	86.5	46.9	109	85
Colombia	2.2	79	72	22	483	2576	82.5	90.7	58.5	370	78
Comoros	4.4	47	64	66	713	1764	58.5	49.3	64.3	21	26
Congo	4.5	207	53	79	716	2236	45	78.4	47.3	56	44
Cook Islands	2.6	.	.	29	570	.	97.5	.	.	436	44
Costa Rica	2.1	69	79	14	439	2749	96.5	95.1	49.8	467	96
Côte d'Ivoire	4.5	131	57	74	559	2588	48.5	38.6	44.6	84	13
Croatia	1.3	14	75	5	578	2597	99	97.1	29	839	.
Cuba	1.5	46	78	9	437	2614	94.5	99.8	40	353	77
Cyprus	1.6	7	79	9	412	3283	100	96.3	29	1973	.
Czech Republic	1.2	11	76	4	559	3028	99.5	99	25.8	980	69
Dem. People's Rep. of Korea	1.9	.	67	40	642	2165	79.5	.	31	1	81
Dem. Rep. of the Congo	6.7	.	46	81	921	1557	35	74.9	44.4	8	21
Denmark	1.8	6	78	4	495	3443	100	99.9	24.7	2379	.
Djibouti	4	24	54	72	862	2182	74	58.4	40	90	23
Dominica	2.1	.	.	20	580	2991	90	94	49	387	50
Dominican Republic	2.8	109	72	40	794	2319	83	87.2	50	333	73
Ecuador	2.6	83	75	34	484	2726	84	89.7	54.4	202	73
Egypt	2.9	41	70	31	891	3376	79	59.4	34.4	208	60
El Salvador	2.7	85	71	37	518	2470	80.5	77.7	52.4	351	73
Equatorial Guinea	5.4	124	50	78	938	.	47	80.5	39	160	.
Eritrea	5.1	70	57	73	686	1669	29	47.6	.	33	8
Estonia	1.5	22	73	5	664	2946	97.5	99.8	36	521	70
Ethiopia	5.3	105	54	82	817	1887	18	35.1	29.8	19	15
Fiji	2.8	34	68	24	767	2778	58.5	91.9	50	160	35
Finland	1.8	12	79	4	405	3169	100	99.9	26.9	1794	.
France	1.9	7	80	6	387	3597	100	99	32.7	2542	75
Gabon	3.1	93	60	68	716	2585	60.5	53.3	41.5	552	33
Gambia	4.8	92	55	72	830	2273	67.5	32.8	50.2	39	18
Georgia	1.4	45	71	25	554	2236	90	99.9	40.8	153	47
Germany	1.4	8	79	5	429	3506	100	99	28.3	2670	75
Ghana	3.9	66	57	73	699	2613	40.5	49.8	40.8	65	24
Greece	1.3	9	79	4	436	3738	98.5	94.2	34.3	1449	.
Grenada	2.3	44	68	26	827	2758	95.5	.	45	388	54
Guatemala	4.2	109	70	51	515	2148	85.5	63.3	53.7	217	43
Guinea	5.5	155	56	77	844	2320	38.5	18.1	43.3	47	9
Guinea-Bissau	7.1	129	47	83	925	2486	44	27.4	35.5	34	10

Guyana	2.3	64	66	41	835	2639	85.5	98.5	43.2	116	34
Haiti	3.6	48	60	67	740	2046	40	51.2	59.5	61	32
Honduras	3.3	95	70	47	761	2394	69	80.2	53.8	138	65
Hungary	1.3	21	73	3	693	3552	99.5	99.3	26.9	852	77
Iceland	2	16	81	4	375	3214	100	99	25	2738	.
India	2.8	70	64	56	713	2489	52.5	47.8	36.8	63	56
Indonesia	2.2	41	70	31	690	2913	64.5	86.8	34.3	37	61
Iran (Islamic Republic of)	2	20	70	28	687	2935	88.5	70.4	43	387	79
Iraq	4.3	82	68	42	1018	.	76	64.2	42	84	50
Ireland	2	17	79	7	459	3701	100	99	34.3	1950	.
Israel	2.8	15	80	9	368	3510	100	95.9	39.2	1845	.
Italy	1.4	5	80	5	372	3663	100	98	36	2061	60
Jamaica	2.5	79	72	35	605	2686	88	91.6	45.5	313	69
Japan	1.3	5	82	8	284	2753	100	99	38.1	1967	56
Jordan	3.1	25	72	29	711	2732	93.5	84.7	37.7	302	57
Kazakhstan	2.3	30	66	25	1145	2386	96.5	99.3	33.9	198	51
Kenya	5	104	53	82	729	2037	46	79.7	42.5	51	39
Kiribati	4.1	.	61	42	730	2910	46	.	.	154	22
Kuwait	2.2	13	78	13	454	3151	.	91	30	903	50
Kyrgyzstan	2.5	32	68	35	1012	2877	87.5	98.1	32.9	62	48
Lao People's Dem. Republic	3.2	40	64	62	828	2303	34	60.9	34.6	41	38
Latvia	1.3	15	71	5	710	2720	88.5	99.8	35.7	456	48
Lebanon	2.2	17	72	20	715	3151	99	82.2	45	801	58
Lesotho	3.4	77	43	86	581	2304	55.5	94.5	52.6	65	37
Liberia	6.8	142	57	84	931	2176	47.5	41.6	.	14	11
Libyan Arab Jamahiriya	2.8	3	74	29	654	3324	84	72	35.8	385	45
Lithuania	1.3	23	71	5	635	3293	.	99.6	36	543	47
Luxembourg	1.7	13	79	7	419	.	100	99.9	26	3137	.
Madagascar	4.8	136	59	74	799	2138	28	62.5	47.5	21	27
Malawi	5.6	140	47	87	796	2166	59	49.8	37.9	38	41
Malaysia	2.6	13	74	28	623	2917	96	85.4	49.2	289	55
Maldives	2.6	14	67	35	953	2552	72.5	96.4	39	170	39
Mali	6.5	163	53	83	967	2358	46.5	39.6	40.1	52	8
Malta	1.4	12	80	6	433	3543	50049.5	93.6	28	2864	.
Marshall Islands	3.8	.	.	34	961	.	84.5	93.7	.	580	45
Mauritania	4.4	91	63	73	812	2762	36	43.4	39	40	9
Mauritius	1.9	39	72	10	731	2989	97	80.5	48.1	302	76
Mexico	2.2	66	74	25	501	3154	84.5	89.6	46.1	507	71
Micronesia (Fed. States of)	3.8	27	68	32	682	.	59	.	.	216	.

Monaco	1.8	.	.	7	321	.	.	99	33	4377	.
Mongolia	1.9	15	66	32	923	2084	58	97.5	32.8	108	66
Morocco	1.8	19	71	39	655	2966	72.5	39.6	39.5	109	63
Mozambique	2.4	155	43	81	777	1939	34	32.7	47.3	21	16
Myanmar	5.2	19	61	56	775	2806	65	.	40	11	34
Namibia	2.1	76	52	82	513	2743	56.5	83.5	74.3	243	55
Nauru	3.2	.	.	24	1093	.	.	.	.	940	36
Nepal	3	104	63	60	769	2446	51.5	34.9	47.3	40	48
Netherlands	3.3	4	79	6	425	3336	100	99	30.9	2337	79
New Zealand	1.7	23	80	5	398	3211	100	99	36.2	1686	75
Nicaragua	2	114	72	39	705	2223	61.5	67.8	43.1	133	72
Niger	2.8	169	56	86	1030	2121	23	15.1	43.9	16	11
Nigeria	7.2	127	47	81	909	2743	38.5	60.6	43.7	59	15
Niue	5.4	.	.	33	595	.	100	.	.	496	23
Norway	1.8	9	80	4	391	3338	100	99.9	25.8	3039	.
Oman	3	11	75	16	664	.	84.5	73.5	32	461	32
Pakistan	3.5	46	65	64	717	2456	68	36	30.6	40	30
Palau	2.5	.	69	29	735	.	77.5	.	25	1046	17
Panama	2.6	84	75	35	417	2215	80.5	91.2	54.9	560	.
Papua New Guinea	3.8	58	57	65	772	2177	41.5	50.9	50.9	64	32
Paraguay	3.1	74	71	33	602	2544	68	93	58.4	309	79
Peru	2.5	56	73	41	534	2599	73	89.4	49.6	232	71
Philippines	3.3	46	71	44	620	2375	81	92.7	44	80	51
Poland	1.2	14	75	4	583	3401	100	99.7	34.9	583	49
Portugal	1.5	17	78	9	456	3757	98	91.3	38.5	1509	.
Qatar	2.7	17	75	17	512	.	100	88.6	39	1259	43
Republic of Korea	1.2	5	78	6	470	3093	50045.5	99	31.6	747	41
Republic of Moldova	1.4	35	68	10	963	2628	85	98.6	35.6	86	68
Romania	1.3	32	72	9	706	3329	79	96.3	31.5	320	70
Russian Federation	1.3	26	65	8	904	2918	91.5	99.2	39.9	410	.
Rwanda	5.9	38	48	83	878	2058	45	64.7	46.8	24	36
Saint Kitts and Nevis	2.3	.	.	27	691	3095	97.5	.	42.6	541	54
Saint Lucia	2.2	62	74	17	522	2958	93.5	90.6	42.6	429	47
Saint Vincent and Grenadines	2.2	60	71	31	674	2642	50049.5	99	56	282	.
Samoa	4	30	71	32	766	.	94.5	99.4	.	155	43
San Marino	1.3	.	.	5	357	.	.	.	.	2870	.
Sao Tome and Principe	3.9	69	65	71	788	2484	52	77.9	.	.	30
Saudi Arabia	3.4	27	73	24	678	2837	.	70.8	39.2	692	32

Senegal	4.7	105	55	74	852	2270	49.5	29.2	41.3	54	12
Serbia and Montenegro	1.8	23	73	.	.	2660	96	94.1	30	411	41
Seychelles	1.7	.	72	17	650	2437	50043	92.3	.	742	.
Sierra Leone	6.5	128	46	83	1033	1904	34.5	24.4	42.5	17	8
Singapore	1.3	5	80	12	345	.	.	88.6	42.5	1151	62
Slovakia	1.2	21	74	5	628	2789	100	99.7	25	603	74
Slovenia	1.3	6	78	4	480	3149	.	99.8	28.4	1447	74
Solomon Islands	3.9	44	63	50	694	2221	50.5	.	.	80	27
Somalia	6.1	70	47	72	1148	.	22	25.8	30	.	15
South Africa	2.7	61	51	69	867	2908	73	85.7	57.8	519	60
Spain	1.4	12	81	7	379	3387	100	97.2	34.7	1536	81
Sri Lanka	1.9	30	72	8	681	2345	79	89.1	40.2	99	68
Sudan	4.3	59	57	57	986	2272	51.5	50.5	51	37	8
Suriname	2.4	41	69	31	728	2625	87	87.2	52.9	369	46
Swaziland	3.5	88	46	83	707	2541	54.5	80.8	50.4	207	51
Sweden	1.8	8	81	5	372	3100	100	99	25	2283	.
Switzerland	1.4	6	81	5	360	3435	100	99	33.7	3265	82
Syrian Arab Republic	3.1	64	74	25	679	3052	86.5	73.6	42	159	58
Tajikistan	3.4	29	66	72	884	1716	72.5	99.2	32.6	41	37
TFYR Macedonia	1.4	23	74	6	737	2695	89.5	94.1	39	470	.
Thailand	1.8	39	69	42	516	2459	95	90.5	42.5	172	77
Togo	4.9	67	62	78	818	2281	33.5	46.9	34.4	32	17
Tonga	3.8	23	72	31	658	.	98	99	47	163	23
Trinidad and Tobago	1.6	35	69	26	751	2713	91.5	98	40.3	.	43
Tunisia	1.9	7	74	41	537	3310	85.5	65.3	39.8	271	60
Turkey	2.1	40	71	26	701	3374	90	79.6	43.6	432	73
Turkmenistan	2.5	20	63	48	1100	2715	.	98.3	40.8	.	48
Tuvalu	3	.	.	30	979	.	89.5	.	.	324	31
Uganda	6.5	154	51	80	786	2382	44	57.7	45.7	45	24
Ukraine	1.2	29	68	9	881	2898	96.5	99.2	28	198	67
United Arab Emirates	2.3	17	79	18	410	3333	98.5	81.7	31	1263	28
United Kingdom	1.8	25	79	7	441	3312	100	99	36	1846	84
United Republic of Tanzania	5.2	131	54	79	851	1958	43.5	62.2	34.6	30	26
United States of America	2.1	37	78	9	450	3814	99.5	99	46.2	4570	76
Uruguay	2.1	62	76	12	521	2838	100	98.4	44.9	818	.
Uzbekistan	2.5	13	.	48	880	2286	91.5	99	36.8	83	65
Vanuatu	3.8	49	69	39	749	2583	54.5	74	.	127	38

Venezuela	2.6	90	73	21	441	2360	.	92.7	48.2	510	77
Viet Nam	2.2	17	74	39	611	2498	64	86.9	37.7	75	76
Yemen	5.5	70	62	60	941	2041	54.5	30	33.4	84	28
Zambia	5.2	146	44	85	833	1901	51.5	74.8	50.8	52	41
Zimbabwe	3.2	67	43	85	816	2104	62.5	95.7	54	1	60

Figure 1. Model 1. Numbers associated with single-headed arrows are standardized path coefficients. Numbers associated with curved double-headed arrows are correlations. Numbers within circles are the residuals associated with a dependent variable. All path coefficients are significant at the $p<.05$ level.

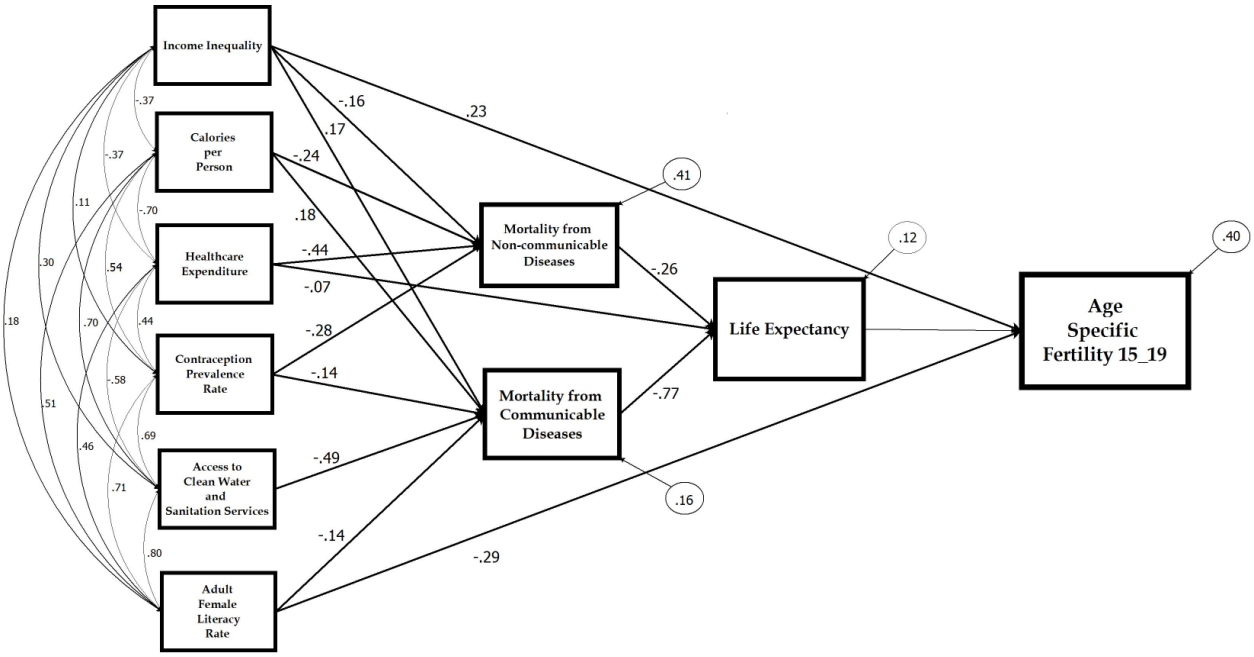

Figure 2. Model 2. Numbers associated with single-headed arrows are standardized path coefficients. Numbers associated with curved double-headed arrows are correlations. Numbers within circles are the residuals associated with a dependent variable. All path coefficients are significant at the $p<.05$ level with the exception of the path between total fertility rate and adult female literacy rate, which was approaching significance at $p=.077$.